```
=> e schofield louis/au
E1          30     SCHOFIELD LORRAINE/AU
E2           6     SCHOFIELD LORRAINE M/AU
E3         144 --> SCHOFIELD LOUIS/AU
E4           3     SCHOFIELD LOUISE/AU
E5           6     SCHOFIELD LYN/AU
E6         150     SCHOFIELD M/AU
E7          44     SCHOFIELD M A/AU
E8           5     SCHOFIELD M G/AU
E9           2     SCHOFIELD M H/AU
E10         59     SCHOFIELD M J/AU
E11          3     SCHOFIELD M L A/AU
E12          1     SCHOFIELD M N/AU

=> s e3
L1         144 "SCHOFIELD LOUIS"/AU

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          69 DUP REM L1 (75 DUPLICATES REMOVED)

=> s l2 and (plasmodium or malaria)
L3          62 L2 AND (PLASMODIUM OR MALARIA)

=> s l3 and (GPI or inositolglycan)
L4          22 L3 AND (GPI OR INOSITOLGLYCAN)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 22 ANSWERS - CONTINUE? Y/(N):y

L4   ANSWER 1 OF 22  BIOSIS  COPYRIGHT (c) 2008 The Thomson Corporation  on STN
AN   2008:243537  BIOSIS
DN   PREV200800243138
TI   Cellular correlates of immunity and risk of disease in semi-immune Papua
     New Guinean children.
AU   Robinson, Leanne J. [Reprint Author]; D'Ombrain, Marthe C.; Stanisic,
     Danielle I.; Bernard, Nicholas; Taraika, Jack; Beeson, James G.; Michon,
     Pascal; King, Chris L.; Mueller, Ivo; Schofield, Louis
CS   Walter and Eliza Hall Inst Med Res, Melbourne, Vic 3050, Australia
SO   International Journal for Parasitology, (JAN 2008) Vol. 38, No. Suppl. 1,
     pp. S29.
     Meeting Info.: 3rd Molecular Approaches to Malaria Meeting (MAM 2008).
     Lorne, AUSTRALIA. February 03 -07, 2008. BioMalPar; Boehringer Ingelheim
     Foods; Burroughs Wellcome Fund; Fdn Natl Inst Hlth; PATH Malaria Vaccine
     Initiative; Walter & Eliza Hall Inst Med Res; Wellcome Trust; ARC/NHMRC
     Net Parasitol; Australian Soc Biochem & Molecular Biol; Lorne Protein
     Conf; GlaxoSmithKline.
     CODEN: IJPYBT. ISSN: 0020-7519.
DT   Conference; (Meeting)
     Conference; Abstract; (Meeting Abstract)
LA   English
ED   Entered STN: 2 Apr 2008
     Last Updated on STN: 2 Apr 2008
AU.  .  .  D'Ombrain, Marthe C.; Stanisic, Danielle I.; Bernard, Nicholas;
     Taraika, Jack; Beeson, James G.; Michon, Pascal; King, Chris L.; Mueller,
     Ivo; Schofield, Louis
IT   .  .  .
        cell: immune system, blood and lymphatics; gamma delta T cells: immune
        system; alpha beta T cell: immune system
IT   Diseases
           malaria: blood and lymphatic disease, parasitic disease
```

Malaria (MeSH)
IT Chemicals & Biochemicals
IFN-gamma [interferon-gamma]; IL-10 [interleukin-10]; IL-6 [interleukin-6]; IL-2 [interleukin-2]; IL-4 [interleukin-4]; TNF [tumor necrosis factor]; GPI; PfEMP-1
ORGN . . .
child
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum (species): parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 2 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:593264 BIOSIS
DN PREV200700594839
TI The role of leuocytes hearing natural killer complex receptors and killer Immunoglobulin-like receptors in the immunology of malaria.
AU Hansen, Diana S.; D'Ombrain, Marthe C.; Schofield, Louis
[Reprint Author]
CS Royal Melbourne Hosp, Walter and Eliza Hall Inst Med Res, 1G Royal Parade, Parkville, Vic 3050, Australia
schofield@wehi.edu.au
SO Current Opinion in Immunology, (AUG 2007) Vol. 19, No. 4, pp. 416-423.
CODEN: COPIEL. ISSN: 0952-7915.
DT Article
LA English
ED Entered STN: 28 Nov 2007
Last Updated on STN: 28 Nov 2007
AB The biology of Natural Killer (NK) cells and other NK Receptor (NKR)(+) leukocytes has largely been elucidated in viral or cancer systems, and involvement in other diseases or infectious states is less clearly defined. Recently, however, clear evidence has emerged for a role in malaria. NK cells and NKR+ leukocytes significantly control susceptibility and resistance to both malaria infection and severe disease syndromes in murine models, in dependence upon receptors encoded within the Natural Killer Complex (NKC). Plasmodium falciparum can rapidly activate human NKR+ gamma delta T cells and NK cells in vitro, and these responses are controlled partly by NKR loci encoded within the human syntenic NKC and Killer Immunoglobulin-like Receptor (KIR) genomic regions. Neither erythrocytes nor malaria parasites express HLA or MHC Class I-like homologues, or obvious stress-type ligands, suggesting the possibility of novel NKR recognition mechanisms. Parasite-derived ligands such as P. falciparum Erythrocyte Membrane Protein-1 (PfEMP-1) and glycosylphosphatidylinositol (GPI) regulate some of these diverse responses. Population-based immunogenetic analyses should allow the identification of NKC and KIR loci controlling innate and adaptive immune responses to malaria and associated with altered risk of infection and disease.
TI The role of leuocytes hearing natural killer complex receptors and killer Immunoglobulin-like receptors in the immunology of malaria.
AU Hansen, Diana S.; D'Ombrain, Marthe C.; Schofield, Louis
[Reprint Author]
AB. . . in other diseases or infectious states is less clearly defined. Recently, however, clear evidence has emerged for a role in malaria. NK cells and NKR+ leukocytes significantly control

susceptibility and resistance to both malaria infection and severe disease syndromes in murine models, in dependence upon receptors encoded within the Natural Killer Complex (NKC). Plasmodium falciparum can rapidly activate human NKR+ gamma delta T cells and NK cells in vitro, and these responses are controlled. . . partly by NKR loci encoded within the human syntenic NKC and Killer Immunoglobulin-like Receptor (KIR) genomic regions. Neither erythrocytes nor malaria parasites express HLA or MHC Class I-like homologues, or obvious stress-type ligands, suggesting the possibility of novel NKR recognition mechanisms. Parasite-derived ligands such as P. falciparum Erythrocyte Membrane Protein-1 (PfEMP-1) and glycosylphosphatidylinositol (GPI) regulate some of these diverse responses. Population-based immunogenetic analyses should allow the identification of NKC and KIR loci controlling innate and adaptive immune responses to malaria and associated with altered risk of infection and disease.

IT . . .
lymphatics; natural killer cell: immune system, blood and lymphatics; natural killer T cells: immune system, blood and lymphatics

IT Diseases
malaria: blood and lymphatic disease, parasitic disease, immunology
Malaria (MeSH)

IT Chemicals & Biochemicals
glycosylphosphatidylinositol; killer immunoglobulin-like receptors; erythrocyte membrane protein-1; natural killer complex receptors

ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGN Classifier
Sporozoa 35400

Super Taxa
Protozoa; Invertebrata; Animalia

Organism Name
Plasmodium falciparum (species): parasite

Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 3 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2006:582619 BIOSIS

DN PREV200600575816

TI Fatty acids from Plasmodium falciparum down-regulate the toxic activity of malaria glycosylphosphatidylinositols.

AU Debierre-Grockiego, Francoise [Reprint Author]; Schofield, Louis ; Azzouz, Nahid; Schmidt, Jorg

CS Inst Virol, AG Parasitol, Hans Meerwein Str 2, D-35043 Marburg, Germany
debierre@staff.uni-marburg.de

SO Infection and Immunity, (OCT 2006) Vol. 74, No. 10, pp. 5487-5496.
CODEN: INFIBR. ISSN: 0019-9567.

DT Article

LA English

ED Entered STN: 1 Nov 2006
Last Updated on STN: 1 Nov 2006

AB Plasmodium falciparum malaria kills roughly 2.5 million people, mainly children, annually. Much of this mortality is thought to arise from the actions of a malarial toxin. This toxin, identified as glycosylphosphatidylinositol (GPI), is a major pathogenicity determinant in malaria. A malarial molecule, Pfj, labeled by [H-3]glucosamine like the GPIs, was identified as a non-GPI molecule. Here we show that Pfj is able to down-regulate tumor necrosis factor alpha (TNF-alpha) production induced by the GPI of P. falciparum. Mass spectrometry analysis showed that Pfj

was not a single molecule but represented a number of molecules. Separation methods, such as cation-exchange chromatography and thin-layer chromatography, were used to isolate and identify the following four main fatty acids responsible for the inhibitory effect on TNF-alpha production: myristic, pentadecanoic, palmitic, and palmitoleic acids. This regulatory effect on cytokine production suggests that there is balanced bioactivity for the different categories of malarial lipids.

TI Fatty acids from Plasmodium falciparum down-regulate the toxic activity of malaria glycosylphosphatidylinositols.

AU Debierre-Grockiego, Francoise [Reprint Author]; Schofield, Louis ; Azzouz, Nahid; Schmidt, Jorg

AB Plasmodium falciparum malaria kills roughly 2.5 million people, mainly children, annually. Much of this mortality is thought to arise from the actions of a malarial toxin. This toxin, identified as glycosylphosphatidylinositol (GPI), is a major pathogenicity determinant in malaria. A malarial molecule, Pfj, labeled by [H-3]glucosamine like the GPIs, was identified as a non-GPI molecule. Here we show that Pfj is able to down-regulate tumor necrosis factor alpha (TNF-alpha) production induced by the GPI of P. falciparum. Mass spectrometry analysis showed that Pfj was not a single molecule but represented a number of molecules.. . .

IT Major Concepts
Biochemistry and Molecular Biophysics; Parasitology

IT Diseases
malaria: blood and lymphatic disease, infectious disease, parasitic disease, etiology, mortality
Malaria (MeSH)

IT Chemicals & Biochemicals
palmitic acid; tumor necrosis factor-alpha [TNF-alpha]; glycosylphosphatidylinositol; fatty acid; palmitoleic acid; myristic acid; pentadecanoic acid;. . .

ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum (species): parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 4 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2006:479567 BIOSIS

DN PREV200600475590

TI Identification and stoichiometry of glycosylphosphatidylinositol-anchored membrane proteins of the human malaria parasite Plasmodium falciparum.

AU Gilson, Paul R.; Nebl, Thomas; Vukcevic, Damjan; Moritz, Robert L.; Sargeant, Tobias; Speed, Terence P.; Schofield, Louis; Crabb, Brendan S. [Reprint Author]

CS Walter and Eliza Hall Inst Med Res, 1G Royal Parade, Parkville, Vic 3050, Australia
crabb@wehi.edu.au

SO Molecular & Cellular Proteomics, (JUL 2006) Vol. 5, No. 7, pp. 1286-1299. ISSN: 1535-9476.

DT Article

LA English

ED Entered STN: 20 Sep 2006
Last Updated on STN: 20 Sep 2007

AB Most proteins that coat the surface of the extracellular forms of the human malaria parasite Plasmodium falciparum are attached to the plasma membrane via glycosylphosphatidylinositol (

GPI) anchors. These proteins are exposed to neutralizing antibodies, and several are advanced vaccine candidates. To identify the GPI-anchored proteome of P. falciparum we used a combination of proteomic and computational approaches. Focusing on the clinically relevant blood stage of the life cycle, proteomic analysis of proteins labeled with radioactive glucosamine identified GPI anchoring on 11 proteins (merozoite surface protein (MSP)-1, -2, -4, -5, -10, rhoptry-associated membrane antigen, apical sushi protein, Pf92, Pf38, Pf12, and Pf34). These proteins represent similar to 94% of the GPI-anchored schizont/merozoite proteome and constitute by far the largest validated set of GPI-anchored proteins in this organism. Moreover MSP-1 and MSP- 2 were present in similar copy number, and we estimated that together these proteins comprise approximately two-thirds of the total membrane-associated surface coat. This is the first time the stoichiometry of MSPs has been examined. We observed that available software performed poorly in predicting GPI anchoring on P. falciparum proteins where such modification had been validated by proteomics. Therefore, we developed a hidden Markov model (GPI -HMM) trained on P. falciparum sequences and used this to rank all proteins encoded in the completed P. falciparum genome according to their likelihood of being GPI-anchored. GPI-HMM predicted GPI modification on all validated proteins, on several known membrane proteins, and on a number of novel, presumably surface, proteins expressed in the blood, insect, and/or pre-erythrocytic stages of the life cycle. Together this work identified 11 and predicted a further 19 GPI-anchored proteins in P. falciparum.

TI Identification and stoichiometry of glycosylphosphatidylinositol-anchored membrane proteins of the human malaria parasite Plasmodium falciparum.

AU Gilson, Paul R.; Nebl, Thomas; Vukcevic, Damjan; Moritz, Robert L.; Sargeant, Tobias; Speed, Terence P.; Schofield, Louis; Crabb, Brendan S. [Reprint Author]

AB Most proteins that coat the surface of the extracellular forms of the human malaria parasite Plasmodium falciparum are attached to the plasma membrane via glycosylphosphatidylinositol (GPI) anchors. These proteins are exposed to neutralizing antibodies, and several are advanced vaccine candidates. To identify the GPI-anchored proteome of P. falciparum we used a combination of proteomic and computational approaches. Focusing on the clinically relevant blood stage of the life cycle, proteomic analysis of proteins labeled with radioactive glucosamine identified GPI anchoring on 11 proteins (merozoite surface protein (MSP)-1, -2, -4, -5, -10, rhoptry-associated membrane antigen, apical sushi protein, Pf92, Pf38, Pf12, and Pf34). These proteins represent similar to 94% of the GPI-anchored schizont/merozoite proteome and constitute by far the largest validated set of GPI-anchored proteins in this organism. Moreover MSP-1 and MSP- 2 were present in similar copy number, and we estimated that together. . . is the first time the stoichiometry of MSPs has been examined. We observed that available software performed poorly in predicting GPI anchoring on P. falciparum proteins where such modification had been validated by proteomics. Therefore, we developed a hidden Markov model (GPI-HMM) trained on P. falciparum sequences and used this to rank all proteins encoded in the completed P. falciparum genome according to their likelihood of being GPI-anchored. GPI-HMM predicted GPI modification on all validated proteins, on several known membrane proteins, and on a number of novel, presumably surface, proteins expressed. . . the blood, insect, and/or pre-erythrocytic stages of the life cycle. Together this work identified 11 and predicted a further 19 GPI-anchored proteins in P. falciparum.

IT . . .

Parasitology; Mathematical Biology (Computational Biology)
IT Parts, Structures, & Systems of Organisms
blood: blood and lymphatics; plasma membrane
IT Diseases
malaria: blood and lymphatic disease, infectious disease, parasitic disease
Malaria (MeSH)
IT Chemicals & Biochemicals
glycosylphosphatidylinositol; rhoptry-associated membrane antigen; merozoite surface protein-1 [MSP-1]: expression; glucosamine: radioactive; merozoite surface protein-2 [MSP-2]:. . .
ORGN . . .
host
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum (species): parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 5 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2006:98259 BIOSIS
DN PREV200600096187
TI Distinct protein classes including novel merozoite surface antigens in raft-like membranes of Plasmodium falciparum.
AU Sanders, Paul R.; Gilson, Paul R.; Cantin, Greg T.; Greenbaum, Doron C.; Nebl, Thomas; Carucci, Daniel J.; McConville, Malcolm J.; Schofield, Louis; Hodder, Anthony N.; Yates, John R. III; Crabb, Brendan S. [Reprint Author]
CS Walter and Eliza Hall Inst Med Res, 1G Royal Parade, Parkville, Vic 3050, Australia
crabb@wehi.edu.au
SO Journal of Biological Chemistry, (DEC 2 2005) Vol. 280, No. 48, pp. 40169-40176.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 1 Feb 2006
Last Updated on STN: 20 Sep 2007
AB Glycosylphosphatidylinositol (GPI)-anchored proteins coat the surface of extracellular Plasmodium falciparum merozoites, of which several are highly validated candidates for inclusion in a blood-stage malaria vaccine. Here we determined the proteome of gradient-purified detergent-resistant membranes of mature blood-stage parasites and found that these membranes are greatly enriched in GPI-anchored proteins and their putative interacting partners. Also prominent in detergent-resistant membranes are apical organelle (rhoptry), multimembrane-spanning, and proteins destined for export into the host erythrocyte cytosol. Four new GPI-anchored proteins were identified, and a number of other novel proteins that are predicted to localize to the merozoite surface and/or apical organelles were detected. Three of the putative surface proteins possessed six-cysteine (Cys6) motifs, a distinct fold found in adhesive surface proteins expressed in other life stages. All three Cys6 proteins, termed Pf12, Pf38, and Pf41, were validated as merozoite surface antigens recognized strongly by antibodies present in naturally infected individuals. In addition to the merozoite surface, Pf38 was particularly prominent in the

secretory apical organelles. A different cysteine-rich putative GPI-anchored protein, Pf92, was also localized to the merozoite surface. This insight into merozoite surfaces provides new opportunities for understanding both erythrocyte invasion and anti-parasite immunity.

TI Distinct protein classes including novel merozoite surface antigens in raft-like membranes of Plasmodium falciparum.

AU . . Sanders, Paul R.; Gilson, Paul R.; Cantin, Greg T.; Greenbaum, Doron C.; Nebl, Thomas; Carucci, Daniel J.; McConville, Malcolm J.; Schofield, Louis; Hodder, Anthony N.; Yates, John R. III; Crabb, Brendan S. [Reprint Author]

AB Glycosylphosphatidylinositol (GPI)-anchored proteins coat the surface of extracellular Plasmodium falciparum merozoites, of which several are highly validated candidates for inclusion in a blood-stage malaria vaccine. Here we determined the proteome of gradient-purified detergent-resistant membranes of mature blood-stage parasites and found that these membranes are greatly enriched in GPI-anchored proteins and their putative interacting partners. Also prominent in detergent-resistant membranes are apical organelle (rhoptry), multimembrane-spanning, and proteins destined for export into the host erythrocyte cytosol. Four new GPI-anchored proteins were identified, and a number of other novel proteins that are predicted to localize to the merozoite surface and/or. . . individuals. In addition to the merozoite surface, Pf38 was particularly prominent in the secretory apical organelles. A different cysteine-rich putative GPI-anchored protein, Pf92, was also localized to the merozoite surface. This insight into merozoite surfaces provides new opportunities for understanding both. . .

IT Major Concepts
Pharmacology; Biochemistry and Molecular Biophysics

IT Parts, Structures, & Systems of Organisms
erythrocyte: blood and lymphatics

IT Diseases
malaria: blood and lymphatic disease, parasitic disease, prevention and control
Malaria (MeSH)

IT Chemicals & Biochemicals
proteome; glycosylphosphatidylinositol-anchored proteins; merozoite surface protein; malaria vaccine: immunologic-drug, immunostimulant-drug

ORGN Classifier
Sporozoa 35400

Super Taxa
Protozoa; Invertebrata; Animalia

Organism Name
Plasmodium falciparum (species): parasite

Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 6 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2005:430293 BIOSIS

DN PREV200510231401

TI Influence of glycosylphosphatidylinositol anchorage on the efficacy of DNA vaccines encoding Plasmodium yoelii merozoite surface protein 4/5.

AU Wang, Lina [Reprint Author]; Kedzierski, Lukasz; Schofield, Louis ; Coppel, Ross L.

CS Monash Univ, Dept Microbiol, Clayton, Vic 3800, Australia
lina.wang@med.monash.edu.au

SO Vaccine, (JUL 14 2005) Vol. 23, No. 32, pp. 4120-4127.
CODEN: VACCDE. ISSN: 0264-410X.

DT Article

LA English
ED Entered STN: 26 Oct 2005
Last Updated on STN: 26 Oct 2005
AB Immune responses induced to DNA vaccination vary considerably and depend on a variety of factors, including the physical form in which the antigen is expressed by target cells and presented to the immune system. Data on the effect of these factors will aid improved design of DNA vaccines and facilitate their further development. We examined the effect of different forms of surface anchoring on the immunogenicity of a DNA vaccine. A number of constructs were generated encoding Plasmodium yoelii merozoite surface protein 4/5 (PyMSP4/5) with or without its C-terminal glycosylphosphatidylinositol (GPI) attachment signal, replacing the endogenous GPI signal of PyMSP4/5 with that of mouse decay-accelerating factor (DAF), a well-established model for GPI -anchoring in mammalian cells, or the transmembrane anchor and cytoplasmic tail of mouse tissue factor (TF). All constructs were demonstrated to express the full-length PyMSP4/5 in transfected COS cells and induce PyMSP4/5-specific antibodies in mice. The GPI attachment signal of PyMSP4/5 was found to function poorly in mammalian cells and result in a much lower level of PyMSP4/5 expression in vitro than its mammalian counterpart. The DNA vaccine containing the mammalian GPI attachment signal induced the highest levels of antibodies and impacted Ig isotype distribution, consistent with the presence of a CD1-restricted pathway of Ig formation to GPI-anchored membrane proteins. Despite the induction of specific antibodies, none of these DNA vaccines induced sufficient levels of antibodies to protect nice against a lethal challenge with P yoelii. (c) 2005 Elsevier Ltd. All rights reserved.
TI Influence of glycosylphosphatidylinositol anchorage on the efficacy of DNA vaccines encoding Plasmodium yoelii merozoite surface protein 4/5.
AU Wang, Lina [Reprint Author]; Kedzierski, Lukasz; Schofield, Louis ; Coppel, Ross L.
AB. . . of different forms of surface anchoring on the immunogenicity of a DNA vaccine. A number of constructs were generated encoding Plasmodium yoelii merozoite surface protein 4/5 (PyMSP4/5) with or without its C-terminal glycosylphosphatidylinositol (GPI) attachment signal, replacing the endogenous GPI signal of PyMSP4/5 with that of mouse decay-accelerating factor (DAF), a well-established model for GPI-anchoring in mammalian cells, or the transmembrane anchor and cytoplasmic tail of mouse tissue factor (TF). All constructs were demonstrated to express the full-length PyMSP4/5 in transfected COS cells and induce PyMSP4/5-specific antibodies in mice. The GPI attachment signal of PyMSP4/5 was found to function poorly in mammalian cells and result in a much lower level of PyMSP4/5 expression in vitro than its mammalian counterpart. The DNA vaccine containing the mammalian GPI attachment signal induced the highest levels of antibodies and impacted Ig isotype distribution, consistent with the presence of a CD1-restricted pathway of Ig formation to GPI-anchored membrane proteins. Despite the induction of specific antibodies, none of these DNA vaccines induced sufficient levels of antibodies to protect. . .
IT . . .
and Homeostasis)
IT Chemicals & Biochemicals
antibody; tissue factor; glycosylphosphatidylinositol anchor; decay-accelerating factor [DAF]; merozoite surface protein 4/5; C-terminal glycosylphosphatidylinositol [GPI]; DNA vaccine: immunologic-drug, immunostimulant-drug, immunogenicity, vaccine
ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium yoelii (species): parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 7 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2003:458136 BIOSIS
DN PREV200300458136
TI CD1d-restricted NKT cells contribute to malarial splenomegaly and enhance parasite-specific antibody responses.
AU Hansen, Diana S. [Reprint Author]; Siomos, Mary-Anne; de Koning-Ward, Tania; Buckingham, Lynn; Crabb, Brendan S.; Schofield, Louis
CS The Walter and Eliza Hall Institute of Medical Research, 1G Royal Parade, Parkville, Victoria, 3050, Australia
hansen@wehi.edu.au
SO European Journal of Immunology, (September 2003) Vol. 33, No. 9, pp. 2588-2598. print.
ISSN: 0014-2980 (ISSN print).
DT Article
LA English
ED Entered STN: 8 Oct 2003
Last Updated on STN: 8 Oct 2003
AB CD1d-restricted NKT cells are a novel T cell lineage with unusual features. They co-express some NK cell receptors and recognize glycolipid antigens through an invariant T cell receptor (TCR) in the context of CD1d molecules. Upon activation through the TCR, NKT cells produce large amounts of IFN-gamma and IL-4. It has been proposed that rapid cytokine output by activated NKT cells may induce bystander activation of other lymphoid lineages. The impact of CD1d-restricted NKT cell activation in the induction of B cell-mediated immune responses to infection is still unclear. We show here that CD1-restricted NKT cells contribute to malarial splenomegaly associated with expansion of the splenic B cell pool and enhance parasite-specific antibody formation in response to Plasmodium berghei infection. The increased B cell-mediated response correlates with the ability of NKT cells to promote Th2 immune responses. Additionally, antibody responses against the glycosylphosphatidylinositol (GPI)-anchored protein merozoite surface protein 1 (MSP-1) were found to be significantly lower in CD1-/- mice compared to wild-type animals. P. berghei-infected MHC class II (MHCII)-/- mice also generated antibodies against MSP-1, suggesting that antibody production against GPI-anchored antigens in response to malaria infection can arise from both MHCII-dependent and independent pathways.
AU Hansen, Diana S. [Reprint Author]; Siomos, Mary-Anne; de Koning-Ward, Tania; Buckingham, Lynn; Crabb, Brendan S.; Schofield, Louis
AB. . . to malarial splenomegaly associated with expansion of the splenic B cell pool and enhance parasite-specific antibody formation in response to Plasmodium berghei infection. The increased B cell-mediated response correlates with the ability of NKT cells to promote Th2 immune responses. Additionally, antibody responses against the glycosylphosphatidylinositol (GPI)-anchored protein merozoite surface protein 1 (MSP-1) were found to be significantly lower in CD1-/- mice compared to wild-type animals. P. berghei-infected MHC class II (MHCII)-/- mice also generated antibodies against MSP-1, suggesting that antibody production against GPI-anchored antigens in response to malaria infection can arise from both MHCII-dependent and independent pathways.

IT . . .
Cell Biology; Immune System (Chemical Coordination and Homeostasis); Infection
IT Parts, Structures, & Systems of Organisms
NKT cells
IT Diseases
malaria infection: infectious disease, parasitic disease
Malaria (MeSH)
IT Diseases
malarial splenomegaly: blood and lymphatic disease, infectious disease, parasitic disease
IT Chemicals & Biochemicals
CD1-d; IFN-gamma [interferon-gamma]; IL-4 [interleukin-4]; NK cell receptors; cytokine; glycosylphosphatidylinositol [GPI]; merozoite surface protein 1 [MSP]; parasite-specific

L4 ANSWER 8 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2002:471725 BIOSIS
DN PREV200200471725
TI Synthetic GPI as a candidate antitoxic vaccine in a model of malaria.
AU Schofield, Louis [Reprint author]; Hewitt, Michael C.; Evans, Krystal; Siomos, Mary-Anne; Seeberger, Peter H.
CS Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital, Post Office, Melbourne, VIC, 3050, Australia
schofield@wehi.edu.au; seeberg@mit.edu
SO Nature (London), (15 August, 2002) Vol. 418, No. 6899, pp. 785-789. print.
CODEN: NATUAS. ISSN: 0028-0836.
DT Letter
LA English
ED Entered STN: 11 Sep 2002
Last Updated on STN: 11 Sep 2002
AB The malaria parasite Plasmodium falciparum infects 5-10% of the world's population and kills two million people annually. Fatalities are thought to result in part from pathological reactions initiated by a malarial toxin. Glycosylphosphatidylinositol (GPI) originating from the parasite has the properties predicted of a toxin; however, a requirement for toxins in general and GPI in particular in malarial pathogenesis and fatality remains unproven. As anti-toxic vaccines can be highly effective public health tools, we sought to determine whether anti-GPI vaccination could prevent pathology and fatalities in the Plasmodium berghei/rodent model of severe malaria. The P. falciparum GPI glycan of the sequence NH2-CH2-CH2-PO4-(Manalpha1-2)6Manalpha1-2Manalpha1-6Manalpha-1-4GlcNH2alpha1-6myo-inositol-1,2-cyclic-phosphate was chemically synthesized, conjugated to carriers, and used to immunize mice. Recipients were substantially protected against malarial acidosis, pulmonary oedema, cerebral syndrome and fatality. Anti-GPI antibodies neutralized pro-inflammatory activity by P. falciparum in vitro. Thus, we show that GPI is a significant pro-inflammatory endotoxin of parasitic origin, and that several disease parameters in malarious mice are toxin-dependent. GPI may contribute to pathogenesis and fatalities in humans. Synthetic GPI is therefore a prototype carbohydrate anti-toxic vaccine against malaria.
TI Synthetic GPI as a candidate antitoxic vaccine in a model of malaria.
AU Schofield, Louis [Reprint author]; Hewitt, Michael C.; Evans, Krystal; Siomos, Mary-Anne; Seeberger, Peter H.
AB The malaria parasite Plasmodium falciparum infects 5-10% of the world's population and kills two million people annually.

Fatalities are thought to result in part from pathological reactions initiated by a malarial toxin. Glycosylphosphatidylinositol (GPI) originating from the parasite has the properties predicted of a toxin; however, a requirement for toxins in general and GPI in particular in malarial pathogenesis and fatality remains unproven. As anti-toxic vaccines can be highly effective public health tools, we sought to determine whether anti-GPI vaccination could prevent pathology and fatalities in the Plasmodium berghei/rodent model of severe malaria. The P. falciparum GPI glycan of the sequence NH2-CH2-CH2-PO4-(Manalpha1-2)6Manalpha1-2Manalpha1-6Manalpha-1-4GlcNH2alpha1-6myo-inositol-1,2-cyclic-phosphate was chemically synthesized, conjugated to carriers, and used to immunize mice. Recipients were substantially protected against malarial acidosis, pulmonary oedema, cerebral syndrome and fatality. Anti-GPI antibodies neutralized pro-inflammatory activity by P. falciparum in vitro. Thus, we show that GPI is a significant pro-inflammatory endotoxin of parasitic origin, and that several disease parameters in malarious mice are toxin-dependent. GPI may contribute to pathogenesis and fatalities in humans. Synthetic GPI is therefore a prototype carbohydrate anti-toxic vaccine against malaria.

IT Major Concepts
Parasitology; Pharmacology
IT Diseases
cerebral syndrome: nervous system disease, etiology
IT Diseases
malaria: blood and lymphatic disease, parasitic disease, drug therapy
Malaria (MeSH)
IT Diseases
malarial acidosis: metabolic disease, parasitic disease
IT Diseases
pulmonary edema: respiratory system disease
Pulmonary Edema (MeSH)
IT Chemicals. . .
ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium berghei: parasite
Plasmodium falciparum: parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 9 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2002:452549 BIOSIS
DN PREV200200452549
TI Genes for glycosylphosphatidylinositol toxin biosynthesis in Plasmodium falciparum.
AU Delorenzi, Mauro; Sexton, Adrienne; Shams-Eldin, Hosam; Schwarz, Ralph T.; Speed, Terry; Schofield, Louis [Reprint author]
CS The Walter and Eliza Hall Institute of Medical Research, Melbourne, Victoria, 3050, Australia
schofield@wehi.edu.au
SO Infection and Immunity, (August, 2002) Vol. 70, No. 8, pp. 4510-4522. print.
CODEN: INFIBR. ISSN: 0019-9567.

DT Article
LA English
ED Entered STN: 21 Aug 2002
Last Updated on STN: 21 Aug 2002
AB About 2.5 million people die of Plasmodium falciparum malaria every year. Fatalities are associated with systemic and organ-specific inflammation initiated by a parasite toxin. Recent studies show that glycosylphosphatidylinositol (GPI) functions as the dominant parasite toxin in the context of infection. GPIs also serve as membrane anchors for several of the most important surface antigens of parasite invasive stages. GPI anchoring is a complex posttranslational modification produced through the coordinated action of a multi-component biosynthetic pathway. Here we present eight new genes of P. falciparum selected for encoding homologs of proteins essential for GPI synthesis: PIG-A, PIG-B, PIG-M, PIG-O, GPI1, GPI8, GAA-1, and DPM1. We describe the experimentally verified mRNA and predicted amino acid sequences and in situ localization of the gene products to the parasite endoplasmic reticulum. Moreover, we show preliminary evidence for the PIG-L and PIG-C genes. The biosynthetic pathway of the malaria parasite GPI offers potential targets for drug development and may be useful for studying parasite cell biology and the molecular basis for the pathophysiology of parasitic diseases.
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IT . . .
and Molecular Biophysics); Parasitology
IT Parts, Structures, & Systems of Organisms
T cells: blood and lymphatics, immune system
IT Diseases
malaria: blood and lymphatic disease, parasitic disease
Malaria (MeSH)
IT Chemicals & Biochemicals
glycosylphosphatidylinositol toxin: biosynthesis
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum: parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans
GEN Plasmodium falciparum PIG-C gene (Sporozoa); Plasmodium

falciparum PIG-L gene (Sporozoa)

L4 ANSWER 10 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2000:88912 BIOSIS
DN PREV200000088912
TI Specificity in signal transduction among glycosylphosphatidylinositols of Plasmodium falciparum, Trypanosoma brucei, Trypanosoma cruzi and Leishmania spp.
AU Tachado, Souvenir D.; Mazhari-Tabrizi, Ramin; Schofield, Louis [Reprint author]
CS Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital, Parkville, VIC, 3050, Australia
SO Parasite Immunology (Oxford), (Dec., 1999) Vol. 21, No. 12, pp. 609-617. print.
CODEN: PAIMD8. ISSN: 0141-9838.
DT Article
LA English
ED Entered STN: 10 Mar 2000
Last Updated on STN: 3 Jan 2002
AB Glycosylphosphatidylinositols (GPIs) and related glycoconjugates of parasite origin have been shown to regulate both the innate and acquired immune systems of the host. This is achieved through the activation of novel GPI-dependent signalling pathways in macrophages, lymphocytes and other cell types. Parasite GPIs impart at least two distinct signals to host cells through the structurally distinct inositolphosphoglycan (IPG) and fatty acid domains. Binding of IPG to as yet uncharacterized cell surface receptor(s) leads to activation of src-family protein tyrosine kinases: depending upon structure, GPI -derived fatty acids can either activate or antagonize protein kinase C, and may enter the sphingomyelinase pathway. The degree of fatty acid saturation may also contribute to signalling activity. Thus, variation in structure of parasite GPIs imparts different properties of signal transduction upon this class of glycolipid. The divergent activities of GPIs from various protozoal taxa reflect global aspects of the host/parasite relationship, suggesting that GPI signalling is a central determinant of disease in malaria, leishmaniasis and both American and African trypanosomiases.
TI Specificity in signal transduction among glycosylphosphatidylinositols of Plasmodium falciparum, Trypanosoma brucei, Trypanosoma cruzi and Leishmania spp.
AU Tachado, Souvenir D.; Mazhari-Tabrizi, Ramin; Schofield, Louis [Reprint author]
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IT . . .
lymphatics, immune system; macrophage: blood and lymphatics, immune system
IT Diseases
leishmaniasis: integumentary system disease, parasitic disease

Leishmaniasis (MeSH)
IT Diseases
malaria: blood and lymphatic disease, parasitic disease
Malaria (MeSH)
IT Chemicals & Biochemicals
African trypanosomiase; American trypanosomiase; glycolipid; glycosylphosphatidylinositol [GPI]; inositolphosphoglycan [IPG]; protein kinase C: activation; src-family protein tyrosine kinases

L4 ANSWER 11 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1999:87812 BIOSIS
DN PREV199900087812
TI CD1d-restricted immunoglobulin G formation to GPI-anchored antigens mediated by NKT cells.
AU Schofield, Louis [Reprint author]; McConville, Malcolm J.; Hansen, Diana; Campbell, A. Stewart; Fraser-Reid, Bert; Grusby, Michael J.; Tachado, Souvenir D.
CS Walter and Eliza Hall Inst. Med. Res., Post Office, R. Melbourne Hosp., Victoria 3050, Australia
SO Science (Washington D C), (Jan. 8, 1999) Vol. 283, No. 5399, pp. 225-229. print.
CODEN: SCIEAS. ISSN: 0036-8075.
DT Article
LA English
ED Entered STN: 1 Mar 1999
Last Updated on STN: 1 Mar 1999
AB Immunoglobulin G (IgG) responses require major histocompatibility complex (MHC)-restricted recognition of peptide fragments by conventional CD4+ helper T cells. Immunoglobulin G responses to glycosylphosphatidylinositol (GPI)-anchored protein antigens, however, were found to be regulated in part through CD1d-restricted recognition of the GPI moiety by thymus-dependent, interleukin-4-producing CD4+, natural killer cell antigen 1.1 ((NK1.1)+) helper T cells. The CD1-NKT cell pathway regulated immunogobulin G responses to the GPI-anchored surface antigens of Plasmodium and Trypanosoma and may be a general mechanism for rapid, MHC-unrestricted antibody responses to diverse pathogens.
TI CD1d-restricted immunoglobulin G formation to GPI-anchored antigens mediated by NKT cells.
AU Schofield, Louis [Reprint author]; McConville, Malcolm J.; Hansen, Diana; Campbell, A. Stewart; Fraser-Reid, Bert; Grusby, Michael J.; Tachado, Souvenir D.
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IT . . .
blood and lymphatics, immune system, natural killer T cells
IT Chemicals & Biochemicals
circumsporozoite protein: native; immunoglobulin G: CD1d-restricted formation; GPI-anchored antigens
ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,

Rodents, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium: parasite
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 12 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1997:212809 BIOSIS
DN PREV199799519313
TI Signal transduction in macrophages by glycosylphosphatidylinositols of Plasmodium, Trypanosoma, and Leishmania: Activation of protein tyrosine kinases and protein kinase C by inositolglycan and diacylglycerol moieties.
AU Tachado, Souvenir D. [Reprint author]; Gerold, Peter; Schwarz, Ralph; Novakovic, Suzanna; McConville, Malcolm; Schofield, Louis
CS Walter Eliza Hall Inst. Med. Res., VIC 3050, Australia
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 8, pp. 4022-4027.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 22 May 1997
Last Updated on STN: 22 May 1997
AB The perturbation of various glycosylphosphatidylinositol (GPI)-anchored surface proteins imparts profound regulatory signals to macrophages, lymphocytes and other cell types. The specific contribution of the GPI moieties to these events however is unclear. This study demonstrates that purified GPIs of Plasmodium falciparum, Trypanosoma brucei, and Leishmania mexicana origin are sufficient to initiate signal transduction when added alone to host cells as chemically defined agonists. GPIs (10 nM-1 mu-M) induce rapid activation of the protein tyrosine kinase (PTK) p59-hck in macrophages. The minimal structural requirement for PTK activation is the evolutionarily conserved core glycan sequence Man-alpha-1-2Man-alpha-1-6Man-alpha-1-4GlcN1-6myo-inositol. GPI-associated diacylglycerols independently activate the calcium-independent epsilon isoform of protein kinase C. Both signals collaborate in regulating the downstream NF-kappa-B/rel-dependent gene expression of interleukin 1-alpha, tumor necrosis factor (TNF) alpha, and inducible NO synthase. The alkylacyl-glycerol-containing iM4 GIPL of L. mexicana, however, is unable to activate protein kinase C and inhibits TNF expression in response to other agonists, establishing signaling specificity among structurally distinct GPIs. GPI alone appears sufficient to mimic the activities of malaria parasite extracts in the signaling pathway leading to TNF expression. A mAb to GPI blocks TNF induction by parasite extracts indicating that GPI is a necessary agent in this response. As protozoal GPIs are closely related to their mammalian counterparts, the data indicate that GPIs do indeed constitute a novel outside-in signaling system, acting as both agonists and second messenger substrates, and imparting at least two separate signals through the structurally distinct glycan and fatty acid domains. These activities may underlie aspects of pathology and immune regulation in protozoal infections.
TI Signal transduction in macrophages by glycosylphosphatidylinositols of Plasmodium, Trypanosoma, and Leishmania: Activation of protein tyrosine kinases and protein kinase C by inositolglycan and diacylglycerol moieties.

AU Tachado, Souvenir D. [Reprint author]; Gerold, Peter; Schwarz, Ralph; Novakovic, Suzanna; McConville, Malcolm; Schofield, Louis
AB The perturbation of various glycosylphosphatidylinositol (GPI)-anchored surface proteins imparts profound regulatory signals to macrophages, lymphocytes and other cell types. The specific contribution of the GPI moieties to these events however is unclear. This study demonstrates that purified GPIs of Plasmodium falciparum, Trypanosoma brucei, and Leishmania mexicana origin are sufficient to initiate signal transduction when added alone to host cells as. . . kinase (PTK) p59-hck in macrophages. The minimal structural requirement for PTK activation is the evolutionarily conserved core glycan sequence Man-alpha-1-2Man-alpha-1-6Man-alpha-1-4GlcN1-6myo-inositol. GPI -associated diacylglycerols independently activate the calcium-independent epsilon isoform of protein kinase C. Both signals collaborate in regulating the downstream NF-kappa-B/rel-dependent gene. . . activate protein kinase C and inhibits TNF expression in response to other agonists, establishing signaling specificity among structurally distinct GPIs. GPI alone appears sufficient to mimic the activities of malaria parasite extracts in the signaling pathway leading to TNF expression. A mAb to GPI blocks TNF induction by parasite extracts indicating that GPI is a necessary agent in this response. As protozoal GPIs are closely related to their mammalian counterparts, the data indicate. . .
IT Miscellaneous Descriptors
ACTIVATION; BLOOD AND LYMPHATICS; CELL BIOLOGY; ENZYMOLOGY; LEISHMANIA-MEXICANA GLYCOSYLPHOSPHATIDYLINOSITOL; MACROPHAGE; PARASITE; PLASMODIUM-FALCIPARUM GLYCOSYLPHOSPHATIDYLINOSITOL; PROTEIN KINASE C; PROTEIN TYROSINE KINASES; SIGNAL TRANSDUCTION; SIGNAL TRANSDUCTION INITIATOR; STRUCTURE-ACTIVITY RELATIONSHIP; TRYPANOSOMA-BRUCEI GLYCOSYLPHOSPHATIDYLINOSITOL
ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 13 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1996:244187 BIOSIS
DN PREV199698792316
TI Structural analysis of the glycosyl-phosphatidylinositol membrane anchor of the merozoite surface proteins-1 and -2 of Plasmodium falciparum.
AU Gerold, Peter; Schofield, Louis; Blackman, Michael J.; Holder, Anthony A.; Schwarz, Ralph T.
CS Zentrum fuer Hygiene und Med. Mikrobiologie, Philipps-Universitaet Marburg, Robert-Koch Str. 17; 35037 Marburg, Germany
SO Molecular and Biochemical Parasitology, (1996) Vol. 75, No. 2, pp. 131-143.
CODEN: MBIPDP. ISSN: 0166-6851.
DT Article
LA English
ED Entered STN: 28 May 1996
Last Updated on STN: 28 May 1996
AB Plasmodium falciparum accumulates the two merozoite surface

proteins-1 and -2 during schizogony. Both proteins are proposed to be anchored in membranes by glycosyl-phosphatidylinositol membrane anchors. In this report the identity of these GPI-anchors is confirmed by labelling with tritiated precursors and additionally by specific enzymatic and chemical treatments. Detailed structural analysis of the core-glycans showed that the GPI-anchors of both proteins possess an extra alpha-1-2 linked mannose at the conserved trimannosyl-core-glycan. MSP-1 and MSP-2 labelled with tritiated myristic acid possess primarily radioactive myristic acid at inositol rings in both GPI-anchors. Additionally the hydrophobic fragments released from (3H)myristic acid labelled GPI-anchors were identified as diacyl-glycerols, carrying preferentially (3H)palmitic acid in an ester-linkage.

TI Structural analysis of the glycosyl-phosphatidylinositol membrane anchor of the merozoite surface proteins-1 and -2 of Plasmodium falciparum.

AU Gerold, Peter; Schofield, Louis; Blackman, Michael J.; Holder, Anthony A.; Schwarz, Ralph T.

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ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 14 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1996:160129 BIOSIS

DN PREV199698732264

TI Glycosylphosphatidylinositol toxin of Plasmodium induces nitric oxide synthase expression in macrophages and vascular endothelial cells by a protein tyrosine kinase-dependent and protein kinase C-dependent signaling pathway.

AU Tachado, Souvenir D. [Reprint author]; Gerold, Peter; McConville, Malcolm J.; Baldwin, Tracey; Quilici, Denis; Schwarz, Ralph T.; Schofield, Louis

CS Immunoparasitol. Unit, Walter and Eliza Hall Inst. Med. Res., Post Office, Royal Melbourne Hosp., Parkville, VIC 3050, Australia

SO Journal of Immunology, (1996) Vol. 156, No. 5, pp. 1897-1907. CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

LA English

ED Entered STN: 11 Apr 1996
Last Updated on STN: 10 Jun 1997

AB In this study, we demonstrate that glycosylphosphatidylinositol (GPI) is a major toxin of Plasmodium falciparum origin responsible for nitric oxide (NO) production in host cells. Purified

malarial GPI is sufficient to induce NO release in a time- and dose-dependent manner in macrophages and vascular endothelial cells, and regulates inducible NO synthase expression in macrophages. GPI -induced NO production was blocked by the NO synthase-specific inhibitor L-N-monomethylarginine. GPI also synergizes with IFN-gamma in regulating NO production. The structurally related molecules dipalmitoylphosphatidylinositol and iM4 glycoinositolphospholipid from Leishmania mexicana had no such activity, and the latter antagonized IFN-gamma-induced NO output. GPI activates macrophages by initiating an early onset tyrosine kinase-mediated signaling process, similar to that induced by total parasite extracts. The tyrosine kinase antagonists tyrphostin and genistein inhibited the release of NO by parasite extracts and by GPI, alone or in combination with IFN-gamma, demonstrating the involvement of one or more tyrosine kinases in the signaling cascade. GPI-induced NO release was also blocked by the protein kinase C inhibitor calphostin C, demonstrating a role for protein kinase C in GPI-mediated cell signaling, and by pyrrolidine dithiocarbamate, indicating the involvement of the NF-kappa-B/c-rel family of transcription factors in cell activation. A neutralizing mAb to malarial GPI inhibited NO production induced by GPI and total malarial parasite extracts in human vascular endothelial cells and murine macrophages, indicating that GPI is a necessary agent of parasite origin in parasite-induced NO output. Thus, in contrast to dipalmitoylphosphatidylinositol and glycoinositolphospholipids of Leishmania, malarial GPI initiates a protein tyrosine kinase- and protein kinase C-mediated signal transduction pathway, regulating inducible NO synthase expression with the participation of NF-kappa-B-rel, which leads to macrophage and vascular endothelial cell activation and downstream production of NO. These events may play a role in the etiology of severe malaria.

TI Glycosylphosphatidylinositol toxin of Plasmodium induces nitric oxide synthase expression in macrophages and vascular endothelial cells by a protein tyrosine kinase-dependent and protein kinase C-dependent. . .

AU Tachado, Souvenir D. [Reprint author]; Gerold, Peter; McConville, Malcolm J.; Baldwin, Tracey; Quilici, Denis; Schwarz, Ralph T.; Schofield, Louis

AB In this study, we demonstrate that glycosylphosphatidylinositol (GPI) is a major toxin of Plasmodium falciparum origin responsible for nitric oxide (NO) production in host cells. Purified malarial GPI is sufficient to induce NO release in a time- and dose-dependent manner in macrophages and vascular endothelial cells, and regulates inducible NO synthase expression in macrophages. GPI -induced NO production was blocked by the NO synthase-specific inhibitor L-N-monomethylarginine. GPI also synergizes with IFN-gamma in regulating NO production. The structurally related molecules dipalmitoylphosphatidylinositol and iM4 glycoinositolphospholipid from Leishmania mexicana had no such activity, and the latter antagonized IFN-gamma-induced NO output. GPI activates macrophages by initiating an early onset tyrosine kinase-mediated signaling process, similar to that induced by total parasite extracts. The tyrosine kinase antagonists tyrphostin and genistein inhibited the release of NO by parasite extracts and by GPI, alone or in combination with IFN-gamma, demonstrating the involvement of one or more tyrosine kinases in the signaling cascade. GPI-induced NO release was also blocked by the protein kinase C inhibitor calphostin C, demonstrating a role for protein kinase C in GPI-mediated cell signaling, and by pyrrolidine dithiocarbamate, indicating the involvement of the NF-kappa-B/c-rel family of transcription factors in cell activation. A neutralizing mAb to malarial GPI inhibited NO production induced by GPI and total malarial parasite extracts in human vascular endothelial cells and murine macrophages, indicating that GPI is

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IT Miscellaneous Descriptors
C-REL; CELL ACTIVATION; CEREBRAL MALARIA; GENE EXPRESSION; INTERFERON-GAMMA; NF-KAPPA-B; NITRIC OXIDE PRODUCTION; PATHOGENESIS; SIGNAL TRANSDUCTION; TRANSCRIPTION FACTOR

ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 15 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1996:160128 BIOSIS

DN PREV199698732263

TI Glycosylphosphatidylinositol toxin of Plasmodium up-regulates intercellular adhesion molecule-1, vascular cell adhesion molecule-1, and E-selectin expression in vascular endothelial cells and increases leukocyte and parasite cytoadherence via tyrosine kinase-dependent signal transduction.

AU Schofield, Louis [Reprint author]; Novakovic, Susanna; Gerold, Peter; Schwarz, Ralph T.; McConville, Malcolm J.; Tachado, Souvenir D.

CS Immunoparasitol. Unit, Walter and Eliza Hall Inst. Med. Res., Post Office, Royal Melbourne Hosp., VIC 3050, Australia

SO Journal of Immunology, (1996) Vol. 156, No. 5, pp. 1886-1896.
CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

LA English

ED Entered STN: 11 Apr 1996
Last Updated on STN: 10 Jun 1997

AB In this study we demonstrate that glycosylphosphatidylinositol (GPI) of malaria parasite origin directly increases cell adhesion molecule expression in purified HUVECs in a dose- and time-dependent manner, resulting in a marked increase in parasite and leukocyte cytoadherence to these target cells. The structurally related glycolipids dipalmitoyl-phosphatidylinositol and iM4 glycoinositolphospholipid of Leishmania mexicana had no such activity. Malarial GPI exerts this effect by activation of an endogenous GPI-based signal transduction pathway in endothelial cells. GPI induces rapid onset tyrosine phosphorylation of multiple intracellular substrates within 1 min of addition to cells in a dose-dependent manner. This activity can be blocked by the protein tyrosine kinase-specific antagonist herbimycin A, genistein, and tyrphostin. These tyrosine kinase antagonists also inhibit GPI -mediated up-regulation of adhesin expression and parasite cytoadherence. GPI-induced up-regulation of adhesin expression and parasite cytoadherence can also be blocked by the NF-kappa-B/c-rel antagonist pyrrolidine-dithiocarbamate, suggesting the involvement of this family of

transcription factors in GPI-induced adhesin expression. The direct activation of endothelial cells by GPI does not require the participation of TNF or IL-1. However, GPI is also responsible for the indirect pathway of increased adhesin expression mediated by TNF and IL-1 output from monocytes/macrophages. Total parasite extracts also up-regulate adhesin expression and parasite cytoadherence in HUVECs, and this activity is blocked by a neutralizing mAb to malarial GPI, suggesting that GPI is the dominant agent of parasite origin responsible for this activity. Thus, a parasite-derived GPI toxin activates vascular endothelial cells by tyrosine kinase-mediated signal transduction, leading to NF-kappa-B/c-rel activation and downstream expression of adhesins, events that may play a central role in the etiology of cerebral malaria

TI Glycosylphosphatidylinositol toxin of Plasmodium up-regulates intercellular adhesion molecule-1, vascular cell adhesion molecule-1, and E-selectin expression in vascular endothelial cells and increases leukocyte and parasite. . .

AU Schofield, Louis [Reprint author]; Novakovic, Susanna; Gerold, Peter; Schwarz, Ralph T.; McConville, Malcolm J.; Tachado, Souvenir D.

AB In this study we demonstrate that glycosylphosphatidylinositol (GPI) of malaria parasite origin directly increases cell adhesion molecule expression in purified HUVECs in a dose- and time-dependent manner, resulting in a. . . to these target cells. The structurally related glycolipids dipalmitoyl-phosphatidylinositol and iM4 glycoinositolphospholipid of Leishmania mexicana had no such activity. Malarial GPI exerts this effect by activation of an endogenous GPI-based signal transduction pathway in endothelial cells. GPI induces rapid onset tyrosine phosphorylation of multiple intracellular substrates within 1 min of addition to cells in a dose-dependent manner.. . . can be blocked by the protein tyrosine kinase-specific antagonist herbimycin A, genistein, and tyrphostin. These tyrosine kinase antagonists also inhibit GPI-mediated up-regulation of adhesin expression and parasite cytoadherence. GPI-induced up-regulation of adhesin expression and parasite cytoadherence can also be blocked by the NF-kappa-B/c-rel antagonist pyrrolidine-dithiocarbamate, suggesting the involvement of this family of transcription factors in GPI-induced adhesin expression. The direct activation of endothelial cells by GPI does not require the participation of TNF or IL-1. However, GPI is also responsible for the indirect pathway of increased adhesin expression mediated by TNF and IL-1 output from monocytes/macrophages. Total. . . also up-regulate adhesin expression and parasite cytoadherence in HUVECs, and this activity is blocked by a neutralizing mAb to malarial GPI , suggesting that GPI is the dominant agent of parasite origin responsible for this activity. Thus, a parasite-derived GPI toxin activates vascular endothelial cells by tyrosine kinase-mediated signal transduction, leading to NF-kappa-B/c-rel activation and downstream expression of adhesins, events that may play a central role in the etiology of cerebral malaria.

IT Miscellaneous Descriptors
C-REL; CEREBRAL MALARIA; GENE EXPRESSION; NF-KAPPA-B; PARASITE-MEDIATED UP-REGULATION; PATHOGENESIS; TRANSCRIPTION FACTOR

ORGN . . .
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia

Organism Name
Plasmodium falciparum
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 16 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1995:79000 BIOSIS
DN PREV199598093300
TI Glycosylphosphatidylinositol toxin of Trypanosoma brucei regulates IL-1-alpha and TNF-alpha expression in macrophages by protein tyrosine kinase mediated signal transduction.
AU Tachado, Souvenir D. [Reprint author]; Schofield, Louis
CS Walter and Eliza Hall Inst. Med. Res., Post Office, Royal Melbourne Hosp., Parkville 3050, Victoria, Australia
SO Biochemical and Biophysical Research Communications, (1994) Vol. 205, No. 2, pp. 984-991.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 22 Feb 1995
Last Updated on STN: 23 Feb 1995
AB A purified, structurally defined glycosylphosphatidylinositol (GPI) derived from the Variant Surface Glycoprotein (VSG) of Trypanosoma brucei, and its biosynthetic precursor P2, was able at submicromolar concentrations to regulate cytokine expression when added directly as pharmacological agonist to host macrophages, by activation of an endogenous protein tyrosine-kinase (PTK) mediated signal transduction pathway. GPI induces rapid onset tyrosine phosphorylation of multiple intracellular substrates, within minutes of addition to LPS-nonresponsive cells, followed shortly thereafter by IL-1-alpha secretion. The PTK antagonists genistein and tyrphostin inhibit both tyrosylphosphorylation and cytokine expression. A monoclonal antibody to GPI also blocks IL-1-alpha induction by total parasite extracts. Thus, as in malaria infection, GPI may induce the cytokine excess causing certain pathological states associated with trypanosomiasis.
AU Tachado, Souvenir D. [Reprint author]; Schofield, Louis
AB A purified, structurally defined glycosylphosphatidylinositol (GPI) derived from the Variant Surface Glycoprotein (VSG) of Trypanosoma brucei, and its biosynthetic precursor P2, was able at submicromolar concentrations. . . added directly as pharmacological agonist to host macrophages, by activation of an endogenous protein tyrosine-kinase (PTK) mediated signal transduction pathway. GPI induces rapid onset tyrosine phosphorylation of multiple intracellular substrates, within minutes of addition to LPS-nonresponsive cells, followed shortly thereafter by IL-1-alpha secretion. The PTK antagonists genistein and tyrphostin inhibit both tyrosylphosphorylation and cytokine expression. A monoclonal antibody to GPI also blocks IL-1-alpha induction by total parasite extracts. Thus, as in malaria infection, GPI may induce the cytokine excess causing certain pathological states associated with trypanosomiasis.

L4 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1993:141666 BIOSIS
DN PREV199395074466
TI Signal transduction in host cells by a glycosylphosphatidylinositol toxin of malaria parasites.
AU Schofield, Louis [Reprint author]; Hackett, Fiona
CS Natl. Inst. Med. Res., The Ridgeway, Mill Hill, London NW7 1AA, UK

SO Journal of Experimental Medicine, (1993) Vol. 177, No. 1, pp. 145-153.
CODEN: JEMEAV. ISSN: 0022-1007.
DT Article
LA English
ED Entered STN: 16 Mar 1993
Last Updated on STN: 17 Mar 1993
AB In this study, we have identified a dominant glycolipid toxin of Plasmodium falciparum. It is a glycosylphosphatidylinositol (GPI). The parasite GPI moiety, free or associated with protein, induces tumor necrosis factor and interleukin 1 production by macrophages and regulates glucose metabolism in adipocytes. Deacylation with specific phospholipases abolishes cytokine induction, as do inhibitors of protein kinase C. When administered to mice in vivo the parasite GPI induces cytokine release, a transient pyrexia, and hypoglycemia. When administered with sensitizing agents it can elicit a profound and lethal cachexia. Thus, the GPI of Plasmodium is a potent glycolipid toxin that may be responsible for a novel pathogenic process, exerting pleiotropic effects on a variety of host cells by substituting for the endogenous GPI-based second messenger/signal transduction pathways. Antibody to the GPI inhibits these toxic activities, suggesting a rational basis for the development of an antiglycolipid vaccine against malaria
.
TI Signal transduction in host cells by a glycosylphosphatidylinositol toxin of malaria parasites.
AU Schofield, Louis [Reprint author]; Hackett, Fiona
AB In this study, we have identified a dominant glycolipid toxin of Plasmodium falciparum. It is a glycosylphosphatidylinositol (GPI). The parasite GPI moiety, free or associated with protein, induces tumor necrosis factor and interleukin 1 production by macrophages and regulates glucose metabolism. . . specific phospholipases abolishes cytokine induction, as do inhibitors of protein kinase C. When administered to mice in vivo the parasite GPI induces cytokine release, a transient pyrexia, and hypoglycemia. When administered with sensitizing agents it can elicit a profound and lethal cachexia. Thus, the GPI of Plasmodium is a potent glycolipid toxin that may be responsible for a novel pathogenic process, exerting pleiotropic effects on a variety of host cells by substituting for the endogenous GPI-based second messenger/signal transduction pathways. Antibody to the GPI inhibits these toxic activities, suggesting a rational basis for the development of an antiglycolipid vaccine against malaria.
ORGN . . .
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
ORGN Classifier
Sporozoa 35400
Super Taxa
Protozoa; Invertebrata; Animalia
Organism Name
Plasmodium falciparum
Taxa Notes
Animals, Invertebrates, Microorganisms, Protozoans

L4 ANSWER 18 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:101015 CAPLUS
DN 140:144698
TI Immunogenic compositions comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria
IN Schofield, Louis

PA The Walter and Eliza Hall Institute of Medical Research, Australia
SO PCT Int. Appl., 134 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004011026	A1	20040205	WO 2003-AU944	20030725
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2493782	A1	20040205	CA 2003-2493782	20030725
	AU 2003245127	A1	20040216	AU 2003-245127	20030725
	AU 2003245127	B2	20071129		
	BR 2003012985	A	20050621	BR 2003-12985	20030725
	EP 1545599	A1	20050629	EP 2003-737755	20030725
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1681529	A	20051012	CN 2003-821710	20030725
	US 20060147476	A1	20060706	US 2005-522494	20050906
	IN 2007DN03027	A	20070817	IN 2007-DN3027	20070423
PRAI	US 2002-398607P	P	20020726		
	WO 2003-AU944	W	20030725		
	IN 2005-DN671	A3	20050221		

AB The present invention relates generally to a method of eliciting or otherwise inducing an immune response to a microorganism and compns. for use therein. More particularly, the present invention relates to a method of inducing an immune response to a parasite utilizing an immunogenic composition comprising a glycosylphosphatidylinositol (referred to herein as ' GPI') inositolglycan domain or its derivative or equivalent The present invention is useful, inter alia, as a prophylactic and/or therapeutic treatment for microorganism infections of mammals such as, for example, parasite infections and in particular infection by Plasmodium species. In another aspect the invention provides a method of diagnosing, monitoring, screening for or otherwise qual. or quant. assessing an immune response to a microorganism and, in particular, a parasite. More particularly, this aspect of the present invention is directed to assessing said immune response utilizing a GPI inositolglycan domain or its derivative or equivalent The development of this aspect of the present invention facilitates, inter alia, the qual. and/or quant. anal. of anti-GPI antibodies in a biol. sample, the identification and/or isolation of unique specificities of antibodies (such as those which bind a parasite derived toxin or the parasite itself), epitope specific screening or the rational design of immunogenic mols. and the generation , thereby, of functionally effective immunointeractive mols.

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Immunogenic compositions comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria

IN Schofield, Louis

AB . . . method of inducing an immune response to a parasite utilizing an

immunogenic composition comprising a glycosylphosphatidylinositol (referred to herein as 'GPI') inositolglycan domain or its derivative or equivalent The present invention is useful, inter alia, as a prophylactic and/or therapeutic treatment for microorganism infections of mammals such as, for example, parasite infections and in particular infection by Plasmodium species. In another aspect the invention provides a method of diagnosing, monitoring, screening for or otherwise qual. or quant. assessing. . . particular, a parasite. More particularly, this aspect of the present invention is directed to assessing said immune response utilizing a GPI inositolglycan domain or its derivative or equivalent The development of this aspect of the present invention
facilitates, inter alia, the qual. and/or quant. anal. of anti-GPI antibodies in a biol. sample, the identification and/or isolation of unique specificities of antibodies (such as those which bind a. . .

ST glycophosphoinositides inositolglycan domain malaria immunogen vaccine antigen immunodiagnosis immunotherapy

IT Antigens
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(MSA-3 (merozoite surface antigen 3); immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Antigens
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(MSA-4 (merozoite surface antigen 4); immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Antigens
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(MSP-2 (merozoite surface protein 2); immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Vaccines
(antimalarial; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Samples
(biol.; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Drug delivery systems
(carriers; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Lipids, biological studies
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(domain; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Diagnosis
(immunodiagnosis; immunogenic compns. comprising inositolglycan

domain of Plasmodium-derived glycophosphoinositide for
diagnosis and therapy against malaria)

IT Epitopes
Immunotherapy
Infection
Malaria
Microorganism
Parasite
Plasmodium (malarial genus)
Plasmodium falciparum
Test kits
Vaccines
(immunogenic compns. comprising inositolglycan domain of
Plasmodium-derived glycophosphoinositide for diagnosis and
therapy against malaria)

IT Antibodies and Immunoglobulins
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of
Plasmodium-derived glycophosphoinositide for diagnosis and
therapy against malaria)

IT Antigens
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of
Plasmodium-derived glycophosphoinositide for diagnosis and
therapy against malaria)

IT MSP-1 (protein)
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of
Plasmodium-derived glycophosphoinositide for diagnosis and
therapy against malaria)

IT Molecules
(immunoreactive; immunogenic compns. comprising inositolglycan
domain of Plasmodium-derived glycophosphoinositide for
diagnosis and therapy against malaria)

IT Oligosaccharides, biological studies
Polysaccharides, biological studies
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inositol; immunogenic compns. comprising inositolglycan
domain of Plasmodium-derived glycophosphoinositide for
diagnosis and therapy against malaria)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; immunogenic compns. comprising inositolglycan
domain of Plasmodium-derived glycophosphoinositide for
diagnosis and therapy against malaria)

IT Glycolipoproteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(phosphatidylinositol-containing, malarial antigen; immunogenic compns.
comprising inositolglycan domain of Plasmodium
-derived glycophosphoinositide for diagnosis and therapy against
malaria)

IT Glycophospholipids

RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(phosphatidylinositol-containing; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Drug design
(rational; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Drug screening
(vaccine; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT Antimalarials
(vaccines; immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

IT 142921-61-7 149864-49-3 154718-48-6 460095-54-9 460095-54-9D, derivs. 653601-83-3D, amino acid derivs. 653601-84-4 653601-85-5D, derivs. 653601-86-6D, derivs. 653601-87-7 653601-88-8D, derivs.
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of Plasmodium-derived glycophosphoinositide for diagnosis and therapy against malaria)

L4 ANSWER 19 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:609398 CAPLUS
DN 137:246241
TI Synthetic GPI as a candidate anti-toxic vaccine in a model of malaria
AU Schofield, Louis; Hewitt, Michael C.; Evans, Krystal; Siomos, Mary-Anne; Seeberger, Peter H.
CS Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital, Victoria, 3050, Australia
SO Nature (London, United Kingdom) (2002), 418(6899), 785-789
CODEN: NATUAS; ISSN: 0028-0836
PB Nature Publishing Group
DT Journal
LA English
AB The malaria parasite Plasmodium falciparum infects 5-10% of the world's population and kills two million people annually. Fatalities are thought to result in part from pathol. reactions initiated by a malarial toxin. Glycosylphosphatidylinositol (GPI) originating from the parasite has the properties predicted of a toxin; however, a requirement for toxins in general and GPI in particular in malarial pathogenesis and fatality remains unproven. As anti-toxic vaccines can be highly effective public health tools, the authors sought to determine whether anti-GPI vaccination could prevent pathol. and fatalities in the P. berghei/rodent model of severe malaria. The P. falciparum GPI glycan of the sequence NH2-CH2-CH2-PO4-(Manα1-2)6Manα1-2Manα1-6Manα1-4GlcNH2α1-6myo-inositol-1,2-cyclic-phosphate was chemical synthesized, conjugated to carriers, and used to immunize mice. Recipients were substantially protected against malarial acidosis, pulmonary edema, cerebral syndrome, and fatality. Anti-GPI antibodies neutralized pro-inflammatory activity by P. falciparum in vitro. Thus, GPI is a pro-inflammatory endotoxin of parasitic origin, and several disease parameters in malarious mice are toxin-dependent. GPI may contribute to pathogenesis and fatalities in humans.

Synthetic GPI is therefore a prototype carbohydrate anti-toxic vaccine against malaria.

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Synthetic GPI as a candidate anti-toxic vaccine in a model of malaria

AU Schofield, Louis; Hewitt, Michael C.; Evans, Krystal; Siomos, Mary-Anne; Seeberger, Peter H.

AB The malaria parasite Plasmodium falciparum infects 5-10% of the world's population and kills two million people annually. Fatalities are thought to result in part from pathol. reactions initiated by a malarial toxin. Glycosylphosphatidylinositol (GPI) originating from the parasite has the properties predicted of a toxin; however, a requirement for toxins in general and GPI in particular in malarial pathogenesis and fatality remains unproven. As anti-toxic vaccines can be highly effective public health tools, the authors sought to determine whether anti-GPI vaccination could prevent pathol. and fatalities in the P. berghei/rodent model of severe malaria. The P. falciparum GPI glycan of the sequence NH2-CH2-CH2-PO4-(Manα1-2)6Manα1-2Manα1-6Manα1-4GlcNH2α1-6myo-inositol-1,2-cyclic-phosphate was chemical synthesized, conjugated to carriers, and used to immunize mice. Recipients were substantially protected against malarial acidosis, pulmonary edema, cerebral syndrome, and fatality. Anti-GPI antibodies neutralized pro-inflammatory activity by P. falciparum in vitro. Thus, GPI is a pro-inflammatory endotoxin of parasitic origin, and several disease parameters in malarious mice are toxin-dependent. GPI may contribute to pathogenesis and fatalities in humans. Synthetic GPI is therefore a prototype carbohydrate anti-toxic vaccine against malaria.

ST glycosylphosphatidylinositol endotoxin prepn malaria vaccine rodent model

IT Vaccines
(antimalarial; synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(endotoxins; synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT Glycophospholipids
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(phosphatidylinositol-containing; synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT Human
Plasmodium berghei
Plasmodium falciparum
(synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT Carbohydrates, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vaccine; synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT Antimalarials
(vaccines; synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT 460095-54-9P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT 97-30-3
RL: RCT (Reactant); RACT (Reactant or reagent)
(synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

IT 83441-60-5P 129163-12-8P 208712-66-7P 439684-07-8P 460095-55-0P 460095-56-1P 460095-57-2P 460095-58-3P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(synthetic glycosylphosphatidylinositol as candidate anti-toxic vaccine in rodent model of malaria)

L4 ANSWER 20 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2000:290843 CAPLUS
DN 132:303491
TI A method of activating T cells with a glycosylphosphatidylinositol, and therapeutic use
IN Schofield, Louis; Hansen, Diana
PA The Walter and Eliza Hall Institute of Medical Research, Australia
SO PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000024406	A1	20000504	WO 1999-AU929	19991027
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1126857	A1	20010829	EP 1999-970921	19991027
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
AU 775222	B2	20040722	AU 2000-11425	19991027
PRAI AU 1998-6758	A	19981027		
WO 1999-AU929	W	19991027		

AB The invention relates generally to a method of activating T cells and more particularly to a method of activating T cells using glycosylphosphatidylinositol (GPI) mols. and derivs. or equivalent thereof. Even more particularly, the method of the invention contemplates a method of activating T cells, using GPI mols. via a CD1-restricted pathway. The method of the invention is useful for a range of therapeutic and/or prophylactic applications including e.g. applications which require skewing of the TH1/TH2 response or which require the induction of antibody production

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IN Schofield, Louis; Hansen, Diana

AB . . . . generally to a method of activating T cells and more particularly to a method of activating T cells using glycosylphosphatidylinositol (GPI) mols. and derivs. or equivalent thereof. Even more particularly, the method of the invention contemplates a method of activating T cells, using GPI mols. via a CD1-restricted pathway. The method of the invention is useful for a range of therapeutic and/or prophylactic applications. . .

IT Antigens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CS (circumsporozoite), GPI complexes; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT MSP-1 (protein)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(GPI complexes; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT Diglycerides
Glycerides, biological studies
Phosphatidylcholines, biological studies
Phosphatidylethanolamines, biological studies
Phosphatidylserines
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(GPI derivs.; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MSP-2 (major merozoite surface protein 2), GPI complexes; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PSA-2, GPI complexes; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT Malaria
Malaria
(cerebral; glycosylphosphatidylinositol for T cell activation, and therapeutic use)
IT Anti-infective agents
Antiarthritics
Antidiabetic agents
Antigen-presenting cell
Antimalarials
Antimicrobial agents
Antitumor agents
B cell (lymphocyte)
CD4-positive T cell
Drug delivery systems
Immunodeficiency
Immunostimulants
Infection
Leishmania mexicana
Neoplasm
Parasiticides
Plasmodium (malarial genus)
Plasmodium berghei
Plasmodium falciparum
Trypanosoma brucei
Vaccines

(glycosylphosphatidylinositol for T cell activation, and therapeutic use)

IT Glycoproteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp63, GPI complexes; glycosylphosphatidylinositol for T cell activation, and therapeutic use)

IT Ovalbumin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(haptenated, GPI conjugates; glycosylphosphatidylinositol for T cell activation, and therapeutic use)

IT Brain, disease
Brain, disease
(malaria; glycosylphosphatidylinositol for T cell activation, and therapeutic use)

IT 56-81-5D, Glycerol, diacyl and alkylacyl and monoalkyl derivs., GPI derivs.
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycosylphosphatidylinositol for T cell activation, and therapeutic use)

L4 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2000:190951 CAPLUS
DN 132:235899
TI Immunogenic compositions and uses thereof
IN Schofield, Louis
PA The Walter and Eliza Hall Institute of Medical Research, Australia
SO PCT Int. Appl., 101 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000015254	A1	20000323	WO 1999-AU770	19990914
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9958420	A	20000403	AU 1999-58420	19990914
	AU 766837	B2	20031023		
	EP 1113815	A1	20010711	EP 1999-945777	19990914
	EP 1113815	B1	20070905		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI	AU 1998-5893	A	19980914		
	WO 1999-AU770	W	19990914		

AB The present invention relates generally to a method of eliciting or otherwise inducing an effective immune response to a micro-organism and compns. for use therein. More particularly, the present invention relates to a method of inducing an immune response to a parasite utilizing an immunogenic composition comprising a glycosylphosphatidylinositol (referred to herein as "GPI") inositolglycan domain or its derivs.

Even more particularly, the present invention contemplates an immunogenic composition comprising the Plasmodium falciparum GPI inositolglycan domain or its derivs. The present invention is useful, inter alia , as a prophylactic and/or therapeutic treatment for disease conditions such as, for example, infection by parasites and in particular infection by Plasmodium species.

RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IN Schofield, Louis

AB . . . method of inducing an immune response to a parasite utilizing an immunogenic composition comprising a glycosylphosphatidylinositol (referred to herein as "GPI") inositolglycan domain or its derivs.
Even more particularly, the present invention contemplates an immunogenic composition comprising the Plasmodium falciparum GPI inositolglycan domain or its derivs. The present invention is useful, inter alia , as a prophylactic and/or therapeutic treatment for disease conditions such as, for example, infection by parasites and in particular infection by Plasmodium species.

ST vaccine Plasmodium falciparum glycosylphosphatidylinositol inositolglycan domain

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MSP-2 (major merozoite surface protein 2); immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Antiserums
Drug delivery systems
Malaria
Mammal (Mammalia)
Microorganism
Parasite
Plasmodium (malarial genus)
Plasmodium falciparum
Vaccines
(immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Antibodies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Antigens
MSP-1 (protein)
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Oligosaccharides, biological studies
Polysaccharides, biological studies
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inositol; immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Antibodies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); THU

(Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(monoclonal; immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT Glycolipoproteins
Glycophospholipids
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(phosphatidylinositol-containing; immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

IT 261757-36-2D, ethanolamine-phosphate derivs.
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunogenic compns. comprising inositolglycan domain of glycosylphosphatidylinositol-anchored antigen for vaccine against microorganism or Plasmodium infection)

L4 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1997:431499 CAPLUS
DN 127:158835
OREF 127:30699a,30702a
TI Glycosyl-phosphatidylinositols in protozoa structure, biosynthesis and intracellular localization
AU Zinecker, Christina; Gerold, Peter; Azzouz, Nahid; Striepen, Boris; Schmidt, Almut; Berhe, Saba; Kimmel, Jurgen; Keddes, Mamdouh H.; Blackman, Michael J.; Schofield, Louis; Ogun, Sola; Damm, Jan B. L.; Melgers, Pedro A. T.; Koolen, Marck; Gerwig, Gerrit J.; Vliegenhardt, Johannes F. G.; Dubremetz, Jean F.; Holder, Anthony A.; Eckert, Volker; Capdeville, Yvonne; Tachado, Souvenir D.; Schwarz, Ralph T.
CS Med. Zentrum fur Hygiene und Med. Mikrobiologie, Philipps-Universitat Marburg, Germany
SO Indian Journal of Biochemistry & Biophysics (1997), 34(1&2), 105-109
CODEN: IJBBBQ; ISSN: 0301-1208
PB National Institute of Science Communication
DT Journal
LA English
AB We are investigating the structure and biosynthesis of glycosyl-phosphatidylinositols (GPI) in the protozoa Toxoplasma gondii, Plasmodium falciparum, Plasmodium yoelii and Paramecium primaurelia. This comparison of structural and biosynthesis data should lead us to common and individual features of the GPI -biosynthesis and transport in different organisms.
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AU Zinecker, Christina; Gerold, Peter; Azzouz, Nahid; Striepen, Boris; Schmidt, Almut; Berhe, Saba; Kimmel, Jurgen; Keddes, Mamdouh H.; Blackman, Michael J.; Schofield, Louis; Ogun, Sola; Damm, Jan B. L.; Melgers, Pedro A. T.; Koolen, Marck; Gerwig, Gerrit J.; Vliegenhardt, Johannes F. G.;. .
AB We are investigating the structure and biosynthesis of glycosyl-phosphatidylinositols (GPI) in the protozoa Toxoplasma gondii, Plasmodium falciparum, Plasmodium yoelii and Paramecium primaurelia. This comparison of structural and biosynthesis data should lead us to common and individual features of the GPI -biosynthesis and transport in different organisms.
IT Paramecium primaurelia
Plasmodium berghei yoelii
Plasmodium falciparum
Protozoa

Toxoplasma gondii
(glycosyl-phosphatidylinositols in protozoa structure, biosynthesis and intracellular localization)